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Distribution of cell junction proteins in the descending colon of *Muc2* mice

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Muc2 mouse strain is an experimental model of inflammatory bowel disease (IBD) lacking Mucin2, which is the prevalent component of intestinal mucus layer. *Muc2* mice exhibit the "leaky gut" syndrome characterized by intestinal barrier disruption, chronic inflammation, diarrhea, bloody stool and increased colorectal cancer risk. Using transmission electron microscopy, we have previously shown that tight junctions (TJ) and adherens junctions (AJ) are impaired in this IBD model. TJ represent the most apical selectively impermeable connections between enterocytes. AJ localize following TJ and serve for cell adhesion and tissue connectivity. Here, we describe the localization of several cell junction proteins in the colon of *Muc2* mice using fluorescent confocal microscopy. As for TJ marker proteins, we used claudin 3, claudin 7, zonula occludens 1 and JAM-A. E-cadherin and β-catenin were used to investigate AJ. Our findings demonstrate the dissociation of claudin 3 from the plasma membrane into cytoplasmic vesicles in the descending colon of mutant mice. Claudin 7 appeared to be less abundant at the apical part of lateral membrane of *Muc2* enterocytes compared to control (p-value < 0.05, $N = 6$). Zonula occludens 1, E-cadherin, β-catenin and JAM-A have showed no significant difference between $Muc2^{+/+}$ and $Muc2^{-/-}$ mice. Previous findings demonstrate that F-actin has impaired dynamics in *Muc2* mice leading to AJ' and TJ' disassembly. We conclude that the increase of intestinal permeability can be at least partially explained by the redistribution of the claudin family proteins possibly due to actin cytoskeleton instability.

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